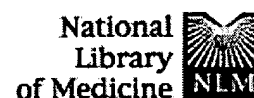


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### Abstract

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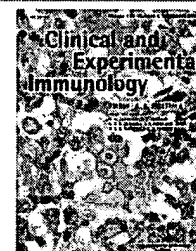
## Evidence of a role for CD200 in regulation of immune rejection of leukaemic tumour cells in C57BL/6 mice

R. M. Gorczynski\*, Z. Chen\*, J. Hu\*, Y. Kai\* & J. Lei\*

Increased expression of the molecule CD200 in mice receiving renal allografts is associated with immunosuppression leading to increased graft survival, and altered cytokine production in lymphocytes harvested from the transplanted animals. Preferential production of IL-4, IL-10 and TGF $\beta$  occurs on donor-specific restimulation *in vitro*, with decreased production of IL-2, IFN $\gamma$  and TNF $\alpha$ . These effects are enhanced by simultaneous infusion of CD200 immunoadhesin (CD200Fc) and donor CD200 receptor (CD200<sup>r</sup>) bearing macrophages to transplanted mice. C57BL/6 mice do not normally resist growth of EL4 or C1498 leukaemia tumour cells. Following transplantation of cyclophosphamide-treated C57BL/6 with T-depleted C3H bone marrow cells, or for the EL4 tumour, immunization of C57BL/6 mice with tumour cells transfected with a vector encoding the co-stimulatory molecule CD80 (EL4-CD80), mice resist growth of tumour challenge. Immunization of C57BL/6 mice with EL4 cells overexpressing CD86 (EL4-CD86) is ineffective. Protection from tumour growth in either model is suppressed by infusion of CD200Fc, an effect enhanced by co-infusion of CD200<sup>r</sup> macrophages. CD200Fc acts on both CD4<sup>+</sup> and CD8<sup>+</sup> cells to produce this suppression. These data are consistent with the hypothesis that immunosuppression following CD200-CD200<sup>r</sup> interaction can regulate a functionally important tumour growth inhibition response in mice.

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